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		<i>DB=PGPB,USPT; PLUR=YES; OP=OR</i>	
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L1 11 (LUTEINIZING HORMONE RECEPTOR OR LHR) AND (HCG OR HUMAN CHORIONIC GONADOTROPIN) AND EXOLOOP

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PROCESSING COMPLETED FOR L1

L2 5 DUP REM L1 (6 DUPLICATES REMOVED)

=> dis his

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L1 11 S (LUTEINIZING HORMONE RECEPTOR OR LHR) AND (HCG OR HUMAN CHORIONIC GONADOTROPIN) AND EXOLOOP  
L2 5 DUP REM L1 (6 DUPLICATES REMOVED)

=> dis ibib abs l2 1-5

L2	ANSWER 1 OF 5	MEDLINE on STN	DUPLICATE 1
ACCESSION NUMBER:	2001269988	MEDLINE	
DOCUMENT NUMBER:	PubMed ID: 10964934		
TITLE:	The role of the hinge region of the luteinizing hormone receptor in hormone interaction and signal generation.		
AUTHOR:	Zeng H; Phang T; Song Y S; Ji I; Ji T H		
CORPORATE SOURCE:	Department of Chemistry, University of Kentucky, Lexington, Kentucky 40506-0055, USA.		
CONTRACT NUMBER:	DK-51469 (NIDDK)		

HD-18702 (NICHD)  
SOURCE: The Journal of biological chemistry, (2001 Feb 2) Vol. 276,  
No. 5, pp. 3451-8. Electronic Publication: 2000-08-29.  
Journal code: 2985121R. ISSN: 0021-9258.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 200106  
ENTRY DATE: Entered STN: 25 Jun 2001  
Last Updated on STN: 5 Jan 2003  
Entered Medline: 21 Jun 2001

AB Luteinizing hormone receptor, a G  
protein-coupled receptor, consists of two halves, the N-terminal  
extracellular hormone binding domain (exodomain) and the C-terminal  
membrane-associated, signal-generating domain (endodomain). The exodomain  
has seven to nine Leu-rich repeats, which are generally thought to form a  
1/3 donut-like structure and interact with human choriogonadotropin (hCG).  
The resulting hCG-exodomain complex adjusts the structure and its association  
with the endodomain, which results in signal generation in the endodomain.  
It is unclear whether the rigid 1/3 donut structure could provide the agility  
and versatility of this dynamic action. In addition, there is no clue as to  
where the endodomain contact point (the signal modulator) in the exodomain is.  
To address these issues, the exodomain was examined by Ala scan and multiple  
substitutions, while receptor peptides were used for photoaffinity labeling  
and affinity cross-linking. Our results show that the C-flanking sequence  
(hinge region), Thr(250)-Gln(268), of the Leu-rich repeats (LRRs) specifically  
interacts with hCG, preferentially hCGalpha. This interaction is inhibited  
by exoloop 2 of the endodomain but not by exoloops 1 and 3, suggesting an  
intimate relationship between Thr(250)-Gln(268), exoloop 2, and hCG. Taken  
together, our observations in this article suggest a new paradigm that the  
LRRs contact the front of hCG, while both flanking regions of the LRRs  
interact with the sides of hCG. This would trap hCG in the 1/3 donut  
structure of the LRRs and enhance the binding affinity. In addition,  
mutations of conserved Ser(255) in the sequence can constitutively activate  
the receptor. This provides a clue for the signal modulator in the exodomain.  
In contrast, a phenyl or phenolic group is necessary at conserved Tyr(253)  
for targeting the receptor to the surface.

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ACCESSION NUMBER: 2003466657 EMBASE  
TITLE: Hormone interactions to Leu-rich repeats in the gonadotropin receptors. III. Photoaffinity labeling of human chorionic gonadotropin with receptor Leu-rich repeat 4 peptide.  
AUTHOR: Jeoung M.; Phang T.; Song Y.S.; Ji I.; Ji T.H.  
CORPORATE SOURCE: T.H. Ji, Dept. of Chemistry, University of Kentucky, Lexington, KY 40506-0055, United States. tji@pop.uky.edu  
SOURCE: Journal of Biological Chemistry, (2 Feb 2001) Vol. 276, No. 5, pp. 3443-3450. .  
Refs: 34  
ISSN: 0021-9258 CODEN: JBCHA3  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 003 Endocrinology  
029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 30 Dec 2003

Last Updated on STN: 30 Dec 2003

AB Human chorionic gonadotropin (hCG) binds to the extracellular N-terminal domain, exodomain, of its receptor, and the resulting hCG-exodomain complex is thought to modulate the membrane associated domain, endodomain, of the receptor to generate hormone signal. The bulk of the exodomain is speculated to assume a crescent structure consisting of eight to nine Leu-rich repeats (LRRs), which may provide the hormone contact sites. Unfortunately, little experimental evidence is available for the precise hormone contact points in the exodomain and the endodomain. The two preceding articles (Song, Y., Ji, I., Beauchamp, J., Isaacs, N., and Ji, T. (2001) J. Biol. Chemical 276, 3426-3435; Song, Y., Ji, I., Beauchamp, J., Isaacs, N., and Ji, T. (2001) J. Biol. Chemical 276, 3436-3442) show that putative LRR2 and LRR4 are crucial for hormone binding. In particular, the N-terminal region of LRR4 assumes the hydrophobic core of the LRR4 loop, whereas the C-terminal region is crucial for signal generation. However, it is unclear whether LRR4 interacts hCG and the endodomain and how it might be involved in signal generation. In this article, our affinity labeling results present the first evidence that the N-terminal region of LRR4 interacts with hCG, preferentially the hCG $\alpha$  subunit and that the hCG/LRR4 complex interacts with exoloop 2 of the endodomain. This interaction offers a mechanism to generate hormone signal.

L2 ANSWER 3 OF 5 MEDLINE on STN DUPLICATE 2  
ACCESSION NUMBER: 1999002809 MEDLINE  
DOCUMENT NUMBER: PubMed ID: 9788760  
TITLE: Surface retention of an inactivating lutropin receptor mutant in exoloop 3.  
AUTHOR: Bhowmick N; Narayan P; Puett D  
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Georgia, Athens 30602, USA.  
CONTRACT NUMBER: DK33973 (NIDDK)  
SOURCE: Molecular and cellular biochemistry, (1998 Oct) Vol. 187, No. 1-2, pp. 221-7.  
Journal code: 0364456. ISSN: 0300-8177.  
PUB. COUNTRY: Netherlands  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
LANGUAGE: English  
FILE SEGMENT: Priority Journals  
ENTRY MONTH: 199812  
ENTRY DATE: Entered STN: 15 Jan 1999  
Last Updated on STN: 15 Jan 1999  
Entered Medline: 18 Dec 1998

AB The heptahelical lutropin receptor (LHR) signals primarily via the Gs-adenylyl cyclase pathway and undergoes ligand-mediated receptor desensitization and internalization. A loss-of-function rat LHR mutant was recently described in which a single amino acid residue replacement in exoloop 3, K583E, had no effect on human choriogonadotropin (hCG) binding but essentially abolished signaling. This LHR mutant is a prime candidate for which to study hCG-mediated receptor internalization since it is highly unlikely that an amino acid residue in exoloop 3, i.e. an extracellular portion of LHR connecting transmembrane helices 6 and 7, could have any direct interaction with Galpha(s), which is located on the cytoplasmic face of the plasma membrane. A method to study endocytosis was adapted that involves concanavalin A binding to the glycoproteins on the cell surface, thus facilitating separation of the plasma membrane fraction from other cellular membrane fractions by sucrose gradient centrifugation. Conditions were used such that a single round of endocytosis could be determined with [125I]hCG. Endocytic rate constants of 0.03 and 0 min<sup>-1</sup> were obtained for LHR and the mutant, respectively, in transfected human embryonic kidney 293 cells;

moreover, internalization of the mutant could not be restored by the addition of 8-Br-cAMP. Thus, the presence of the second messenger cAMP is not sufficient for internalization of ligand-occupied LHR. Rather, it appears that ligand-mediated activation and subsequent internalization of LHR results from an altered conformational state or a conformation-dependent post-ligand binding modification such as phosphorylation.

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ACCESSION NUMBER: 96104114 EMBASE  
DOCUMENT NUMBER: 1996104114  
TITLE: Exoloop 3 of the luteinizing hormone/choriogonadotropin receptor. Lys583 is essential and irreplaceable for human choriogonadotropin (hCG)-dependent receptor activation but not for high affinity hCG binding.  
AUTHOR: Ryu K.-S.; Gilchrist R.L.; Ji I.; Kim S.-J.; Ji T.H.  
CORPORATE SOURCE: Dept. of Molecular Biology, University of Wyoming, Laramie, WY 82071-3944, United States  
SOURCE: Journal of Biological Chemistry, (1996) Vol. 271, No. 13, pp. 7301-7304. .  
ISSN: 0021-9258 CODEN: JBCHA3  
COUNTRY: United States  
DOCUMENT TYPE: Journal; Article  
FILE SEGMENT: 029 Clinical Biochemistry  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 30 Apr 1996  
Last Updated on STN: 30 Apr 1996

AB The luteinizing hormone/choriogonadotropin (CG) receptor belongs to a subfamily of glycoprotein hormone receptors within the seven-transmembrane receptor family. It is comprised of an extracellular N-terminal half of 341 amino acids and a membrane-associated C-terminal half of 303 amino acids. The N-terminal half is capable of high affinity hormone binding whereas the C-terminal half is capable of low affinity hormone binding and receptor activation. However, the precise location of the receptor activation site is currently unknown. We present evidence for the first time that Lys583 of exoloop 3 is crucial and irreplaceable for receptor activation to induce cAMP synthesis. Exoloop 3 is comprised of 11 amino acids and flanked by two Lys residues, Lys573 and Lys583, that are located at the boundaries with the transmembrane columns 6 and 7, respectively. All substitutions including Arg for Lys583 did not affect the high affinity human CG binding, but they resulted in the complete loss of cAMP synthesis induced by human CG. Ala substitutions of the other amino acids in exoloop 3 did not make such a dramatic impact on cAMP induction. The Ala scan revealed two distinct groups of amino acids in terms of their importance in cAMP induction, one group being more important than the other. Interestingly, these two groups of amino acids are arranged in an alternate sequence. This result suggests a specific structure similar to a  $\beta$ -like structure for exoloop 3.

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ACCESSION NUMBER: 97026945 EMBASE  
DOCUMENT NUMBER: 1997026945  
TITLE: Molecular mechanism of LH/CG receptor activation.  
AUTHOR: Ryu K.-S.; Ji I.; Chang L.; Ji T.H.  
CORPORATE SOURCE: T.H. Ji, Department of Molecular Biology, University of Wyoming, Laramie, WY 82071-3944, United States  
SOURCE: Molecular and Cellular Endocrinology, (1996) Vol. 125, No. 1-2, pp. 93-100. .  
Refs: 34

ISSN: 0303-7207 CODEN: MCEND6  
 PUBLISHER IDENT.: S 0303-7207(96)03951-2  
 COUNTRY: Ireland  
 DOCUMENT TYPE: Journal; Conference Article  
 FILE SEGMENT: 003 Endocrinology  
 029 Clinical Biochemistry  
 LANGUAGE: English  
 SUMMARY LANGUAGE: English  
 ENTRY DATE: Entered STN: 15 Feb 1997  
 Last Updated on STN: 15 Feb 1997

AB It is known that the N-terminal half of the LH/CG receptor is responsible for high hCG binding whereas the C-terminal half is capable of receptor activation. Our results suggest that initial hCG binding at the high affinity site in the N-half receptor induces conformational adjustments. This leads to low affinity secondary contacts of the complex of hCG/the N-half receptor with the C-half receptor. This low affinity secondary contact is responsible for activating the receptor. This is based on the following observations. The C-terminal tail of hCG $\alpha$  is known to be involved in activation of the LH/CG receptor. In addition to hCG, we examined the C-terminal three residues (His90-Lys91-Ser92) of the common  $\alpha$  subunit of FSH and TSH. The results show their differential roles in the three hormones. Ser92 is important for binding and cAMP induction of TSH but not for hCG and FSH. Lys91 is important for binding and cAMP induction of hCG, and cAMP induction but not binding of FSH. It is not important for binding or cAMP induction of TSH. His90 is important for all three hormones. When all three residues were truncated, FSH and TSH lose their affinity for binding and cAMP induction, whereas hCG is still capable of binding but not cAMP induction. Therefore, the three amino acids contribute differently in receptor binding and cAMP induction of hCG, FSH and TSH. Our data also indicate that the evolution of the  $\alpha$  subunit has been constrained in order not to impair any one of the hormones. This suggests that each hormone can be independently engineered to improve the potency. To chemically identify the contact site of the  $\alpha$  C-tail of hCG in the LH/CG receptor, a decamer peptide corresponding to the  $\alpha$  subunit sequence from His83 to Ser92 (peptide  $\alpha$ 83-92) was derivatized with UV sensitive reagent, ABG and radio-iodinated. The resulting ABG-125I-peptide  $\alpha$ 83-92 was capable of binding and activating the LH/CG receptor. Furthermore, it specifically photoaffinity-labeled the LH/CG receptor. In addition, the amino group of  $\alpha$ Lys91 of peptide  $\alpha$ 83-92 is crosslinked to a carboxyl group of the receptor, an indication of close association. Reciprocal mutagenesis of  $\alpha$ Lys91 and Asp397 in exoloop 1 of the LH/CG receptor suggests the complementary of this pair in receptor activation but not the high affinity interaction of hCG and the receptor. In addition, Lys583 of exoloop 3 is also crucial for receptor activation. To test the conformational adjustment, ABG was attached to hCG  $\alpha$  and reassociated with untreated  $\beta$  to produce ABG-125I- $\alpha/\beta$ . The extent of inter-subunit crosslinking of ABG-125I- $\alpha/\beta$  bound to the receptor was two to three fold less than unbound ABG-125I- $\alpha/\beta$ . This result indicates structural change at the subunit interface in response to hCG binding to the receptor.

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